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Boundary conditions of otoacoustic emissions

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Chapter 6

Summary and conclusions

In chapters 2 and 3, we studied the postural influences on three types of otoacoustic emissions (OAEs). Persons with normal hearing were tilted in the course of an experiment during which otoacoustic emissions were measured continuously. In virtually all ears, otoacoustic emissions differed between upright and supine posture (with head down 30 degrees). In chapter 2 it was observed that spontaneous otoacoustic emissions (SOAEs) typically show a shift of center frequency (on the order of 10 Hz), together with a change of amplitude (5 dB) and width (5 Hz). These changes, however, occurred in positive as well as negative sense. That is, both up- and downward shifts were observed for all parameters; even within one ear. Chapter 3 shows that the differences of click-evoked otoacoustic emissions (CEOAEs) between both postures could appropriately be described by an amplitude scaling and a time shift. To be exact, due to the downward posture change, CEOAEs showed an amplitude decrease (about 0.5 at 1 kHz) and a phase increase ($\sim$ 80 degrees at 1 kHz), where the latency change was interpreted as a phase shift. For stimulus frequency otoacoustic emissions (SFOAEs), the typical ripple pattern showed a positive shift along the frequency axis on the order of 10 Hz, when the supine position was compared to the upright position. Since this emission's name refers to the frequency at which the emission is elicited, it is rather strange to speak of a frequency shift. When the microphone signal was interpreted as the sum of stimulus and emission, this frequency shift could therefore be apprehended as a positive phase shift of the emission ($\sim$ 90 degrees at 1 kHz). For all three emissions considered, alterations occurred mainly for frequencies below 2 kHz. Experiments from

literature, in which emissions were observed during manipulations of outer- and middle-ear pressure, showed comparable results, suggesting that similar mechanisms are involved (Hauser et al. 1993; Büki et al. 1996).

Changes of posture induce a change of the intracranial pressure (ICP), primarily because of gravity (Chapman et al. 1990). Moreover, since intracranial and intracochlear spaces are connected, pressures in both compartments are closely related (Carlborg et al. 1982). Accordingly, posture changes from upright to supine are thought to cause an increase of the intracranial pressure, and consequently of the intracochlear fluid pressure. As a result of the latter pressure increase, the cochlear windows are thought to bulge outward slightly, which, in turn, causes an alteration of the stiffness of the stapes' annular ligament. Calculations from a middle-ear model in which this stiffness was changed (Büki et al. 1996), showed amplitude and phase changes of middle-ear transmission, similar to the observations mentioned earlier. In effect, an increased stiffness caused a positive phase shift and a small amplitude decrease, mainly for frequencies below 2 kHz. This corresponds well with observations from click-evoked OAEs, stimulus frequency OAEs and distortion product OAEs (e.g., chapter 3; Büki et al. 2000). Spontaneous OAEs showed more complicated changes, but these changes also seem to be in agreement with the model calculations (chapter 2). Thus, OAE measurements can well be tool in monitoring stationary pressure differences of the intracochlear (and intracranial) fluid.

Furthermore, we studied the rate of change of the various otoacoustic emissions, after a posture change. Since our measurements are thought to reflect inner ear pressure — or, actually, pressure changes — the rate of change is probably related to the speed at which pressure changes take place (chapters 2 and 3). After up- and downward postural changes, the time for various emissions to regain stability showed certain differences. For spontaneous OAEs, click-evoked OAEs and distortion product OAEs, the changes after a downward turn occurred more slowly than after an upward turn (see also Büki et al. 2000). Time spans in which the changes occurred were on the order of 10 s; varying from a few seconds to one minute. A measurement of the impedance, or evenly well called a stimulus frequency OAE with fixed frequency, showed no temporal differences after up- and downward turns, however. In this case, both time constants were approximately 11 s.

In chapter 4, the latter experiment was also used to investigate impedance changes in ears of patients with Menière's disease. Here, we did observe a

difference between the time constants of the up- and downward posture changes, where alterations after the downward change were slower again (time constants of 11 versus 15 s). No differences were observed between ears of patients and ears of people with normal hearing, nor between the affected and unaffected — or contralateral — ears of these patients. Thus, the hypothetically disturbed patency of the cochlear aqueduct in patients with Menière's disease is not supported by these findings. It should, however, be noted that intracranial pressure changes after a posture change do not occur instantaneously; the ICP changes were reported to show a rapid and slow component after both sitting up and lying down (Magnæs 1978). Given this complexity of the ICP changes on the one hand, and the diverse time courses we observed on the other, it is questionable to what extent the changes we measured reflect properties of the cochlear aqueduct patency. We conclude that pressure changes due to postural changes are too complicated for studying inner ear pressure dynamics, unless the intracranial pressure is measured simultaneously.

Finally, in chapter 5, click-evoked, as well as distortion product otoacoustic emissions in patients with Menière's disease were analyzed. The incidence of the emissions in affected ears was lower than in unaffected ears. Moreover, the mean emission amplitude in affected ears was lower than in unaffected ears, and, the mean amplitude in unaffected ears was lower than in normal-hearing ears. These differences may well be attributed to the hearing loss involved. Hearing loss did not correlate strongly with emission amplitude; moreover a certain upper boundary for the amplitude was defined by it. Our observations show that also for patients with Menière's disease, otoacoustic emissions are associated with normal or near-normal hearing.